Nutritional aspects of palm oil: an introductory review

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Malaysian palm oil, with its competitive cost of production and versatility, constituted 33% of world trade in oils and fats in 1991. Since >90% of palm oil is used in food applications in more than 90 countries its nutritional characteristics are important. Nutritional research on palm oil in Australia, Europe, the USA, Canada and elsewhere has been published in scientific journals. Key findings are highlighted in this review, showing that palm oil: (a) does not raise blood cholesterol, but often improves LDL/HDL ratio; (b) is not thrombogenic as indicated in both the human and animal studies; (c) is a good alternative to hydrogenated fats avoiding the unfavourable LDL/HDL ratio and increase in Lp(a) they cause and (d) contains carotenoids and tocotrienols which have been shown to have anti-cancer properties for certain types of cancers. (For Malay and Chinese abstracts, see page 224)

Introduction

Although palm oil has been consumed for 5000 years in Africa, where the palm tree originated, its significance as a dietary fat world-wide was not realized until 1984, when it became the largest selling edible oil in the export trade of oils and fats. by 1991 its share of the trade had risen to 33%. More than 90% of palm oil traded is used for food applications, where its versatility and stability are very advantageous. For example, in margarine and shortenings palm oil can stabilize beta prime crystals and this property will ensure a pleasant texture in these products.

As palm oil is mainly used in foodstuffs, interest in nutritional research on the oil naturally arose, and this was further stimulated and accelerated as a result of the anti-palm oil and anti-tropical oil campaign. Several research groups all over the world have examined this problem and their findings have been published in respected international journals such as the *American Journal of Clinical Nutrition* and *Nutrition Research.* There has been a move towards using partially hydrogenated fat in place of palm oil, particularly in the USA. However, recent findings on hydrogenated fat indicate that the trans fatty acids which are formed during the partial hydrogenation of oils and fats may have adverse effects on the human blood lipid profile.

Because of the growing importance of palm oil in the human diet, and also because of the questions arising in regard to the use of hydrogenated fats as an alternative to it, it seemed useful and timely to prepare this summary of recent research findings on palm oil in relation to human nutrition. It differs in style and layout from a formal review, since the intention is to present the essential facts simply and succinctly. The excellent review by Elson[51] provides further reading in greater detail.

The nutritional attributes of palm oil

Palm oil in the codex alimentarius

Palm oil is one of the 16 edible oils possessing an FAO/WHO Food standard under the Codex Alimentarius Commission Programme.

The Codex Alimentarius Commission (Codex 125-1981) comprises 122 member countries. The primary purpose of the Commission programme is to protect the health of consumers and ensure fair practices in the food trade[1].

Palm oil in history

Palm oil has been used as a foodstuff for over 5000 years.

The palm itself is native to West Africa and is a traditional source of food. The oil must also have been traded in ancient times since it was found among grave goods in a 5000 year old tomb at Abydos in Egypt. Chemical analyses of a number of products recovered during the excavation of this tomb were reported in the French journal *Comptes Rendus* in 1897. The analyst, M.C. Friedel, found that one sample of several kilograms, stored in an earthenware jar, had the composition expected of palm oil that had deteriorated during prolonged storage[2].

Palm oil in the contemporary food industry

At present palm oil is consumed world-wide as a cooking oil, and in margarine and shortening, and is also incorporated into fat blends and a wide variety of other food products around the globe[3]. Its versatility is widely appreciated, as shown by trade statistics. Thus about 90% of Malaysia's annual production, currently over 6 million tonnes, is being exported to some 90 countries.

The fatty acid composition of palm oil and its content of natural antioxidants confer exceptional stability at high temperature. This property has been widely recognized[4–8] and as a result palm oil is used for frying industrially and in homes throughout the world.

Fatty acid composition

Palm oil contains an equal proportion of saturated and unsaturated fatty acids, with about 44% palmitic acid, 5% stearic acid, (bpth Saturated), 40% oleic acid (monounsaturated), 10% linoleic acid and 0.4% alpha linolenic acid (both polyunsaturated). Since most commercial palm oil is produced from similar high-yielding hybrids, whatever its source, the chemical composition of different samples is very similar, and is well represented by the round figures just quoted, which are from the *Horticultural Handbook,* as published in the US by the Department ofg Agriculture, Science and Education Administration, an official American publication[9].

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Table 1. Detailed fatty acid composition of palm oil, palm kernel and coconut oils.

Fatty acid	Palm oil	Palm olein	Palm kernel oil	Coconut oil
C6:0			0.3	0.5
C8:0			3.3	7.8
C10:0			3.5	10.0
C12:0	0.1	0.2	47.5	47.5
C14:0	1.0	1.0	16.4	18.1
C16:0	44.3	39.8	8.5	8.8
C16:1	0.1	0.2	–	–
C18:0	4.6	4.4	2.4	2.6
C18:1	38.7	42.5	15.3	6.2
C18:2	10.5	11.2	2.4	1.6
C18:3	0.3	0.4	0.1	–
C20:0	0.3	0.4	0.1	0.1
C20:1	–	–	0.1	–
Number of samples	45		54	21

Distinction between palm oil, palm olein, palm kernel oil and coconut oil

Palm oil and palm olein must be distinguished clearly from palm kernel oil and coconut oil, because they have a very different fatty acid composition, with an appreciably lower level of saturated components, and no significant content of capric, lauric or myristic acids[9].

The oil palm is unique in producing two distinct edible oils from one fruit. Palm oil (and its more liquid fraction, palm olein) is obtained from the flesh of this fruit, while palm kernel oil is obtained from its seed or kernel. The composition of palm kernel oil is similar to that of coconut oil, though it is slightly more unsaturated. The two oils are compared with palm oil and palm olein in Table 1. It will be seen that palm oil is quite different from the two 'lauric' oils.

The figures given in this case are the average of determinations on samples from the main producing areas[10,10a].

Cholesterol is virtually absent from palm oil

The cholesterol content of all vegetable oils, including palm oil, is negligible[11,12]. Before 1960 it was believed that vegetable oils contained no cholesterol at all, but with the advent of highly sensitive analytical methods such as gas chromatography, it was established that, alongside a mixture of plant sterols, predominantly β-sitosterol, minute amounts of cholesterol were also detectable in plant oils.

Table 2 gives typical values for the cholesterol content of a number of crude vegetable oils in parts per million. As can be seen, these values are one or two orders lower than those for the content of cholesterol in animal fats: they are nutritionally insignificant.

Palm oil and the avoidance of trans *fatty acids*

For most food uses palm oil does not require hydrogenation, thus avoiding the formation of the trans fatty acids and uncommon cis-fatty acids found in hydrogenated oils[13].

Palm oil, as a semi-solid fat at 20° C, has natural physical properties which are needed in a number of important food applications. Many other vegetable oils must be partly hydrogenated to attain these properties, and this process generates a range of trans- and uncommon cis fatty acids. Some of these isomers are recognized as having potentially negative effects on fatty acid metabolism and cellular function[13a,13b]. Recent

Table 2. Cholesterol content of crude oils and fats (references 65 and 66) cholesterol (ppm).

Oil	Range	Mean
Coconut	5–24	14
Palm kernel	9–40	17
Sunflower	8–44	17
Palm	13–19	18
Soya	20–35	28
Cotton seed	28–108	44
Rapeseed	25–80	49
Corn	18–95	50
Cocoa butter	–	59
Beef tallow	800–1400	–
Butter	2200–4100	–
Lard	3000–4000	3500

reports from independent research groups show that trans-acids raise plasma LDL cholesterol[14,17–19] and lipoprotein(a)[14–16] in comparison with a diet containing predominantly cis unsaturated acids. (The significance of lipoprotein(a) is discussed later in this article.) In one report[17] the authors concluded that trans fatty acids should be regarded as having an effect 'at least as unfavourable as that of the cholesterol raising fatty acids'.

The data in reference 19 indicate that trans fatty acids are not metabolically equivalent to the natural cis isomers and that they affect the serum lipid profile adversely. A recent epidemiology study involving almost 90 000 women linked trans fatty acid consumption with increased risk of coronary heart disease[19a].

Tocopherols, tocotrienols, carotenoids

Refined palm oil, as used in foods, is a rich source of vitamin E and related substances (tocopherols and tocotrienols, about 500 ppm). Unrefined palm oil is also a rich source of carotenoids[20].

Tocopherols and tocotrienols are natural antioxidants. Also, animal experiments have shown that tocotrienols inhibit the enzyme HMGCoA reductase and consequently the synthesis of cholesterol[21].

The tocopherols and tocotrienols act as scavengers of damaging oxygen-free radicals that have been suggested as playing a role in cellular ageing, atherosclerosis and cancer[22,23]. Laboratory experiments on isolated rat's hearts have shown that a tocopherol/ tocotrienol concentrate from palm oil is more efficient than alpha-tocopherol in protecting the heart against the oxidative injury usually associated with re-perfusion[24].

When the same tocopherol/tocotrienol concentrate was used to treat patients with intermittent claudication in a controlled clinical trial, the test subjects showed a significant increase in walking distance before onset of pain, as compared with the groups given aspirin or a placebo. A measure of oxidation of their serum lipids was also significantly reduced[25].

Low-density lipoproteins (LDLs) are involved in the formation of atherosclerotic lesions, which may be exacerbated when the unsaturated fatty acid components in LDL have become oxidized[25a]. The natural antioxidants protect LDL from oxidation[26].

In a recent cross-cultural epidemiologic study[27] the amount of vitamin E in plasma showed a strong inverse correlation with age-specific mortality from coronary heart disease (CHD). The plasma level of vitamin E appeared to be more important than total plasma cholesterol in explaining cross-cultural differences in CHD mortality. Similar conclusions could not be drawn from studies within a population, however[28, 29]. In a recent paper[30] on lipid peroxidation in skeletal muscle induced by exercise, the authors concluded that there was substantial protection against both resting- and exercise-induced protein oxidation by supplementation with various isomers (alpha-tocopherol, alpha-tocotrienol) of vitamin E.

Unrefined palm oil is a traditional food in West Africa and parts of Brazi., although it is not widely available as a commercial product elsewhere. The main component of its carotenoids is B-carotene, which is a precursor of vitamin A.

The question of whether or not B-carotene has a protective role against some types of cancer is an active area of research[31,32].

Digestibility of palm oil
Like other common edible fats and oils, palm oil is readily digested, absorbed and utilized as a source of energy[33].

Shortages of food fats during and after the First World War led to extensive research into the digestibility of fats, in order to justify the use of unconventional sources. This work has been reviewed[34]. It has been shown repeatedly[33, 35] that fats in general, including palm oil, have a digestibility of 97–99% unless their content of long-chain saturated fatty acids – stearic acid (18:0) and longer—is high. A reduction of digestibility only occurs when the melting point of the fat consumed exceeds 46–48° C.

Palm oil and serum cholesterol
Recent controlled human studies in Europe, the USA and Asia have confirmed that serum total cholesterol does not increase when palm oil is used to replace the major part of other fats in a traditional diet[37–42]. This is in contrast to substitution with the more saturated coconut oil[41, 41a].

The amount of cholesterol in the blood is an indicator of risk for cardiovascular disease[43,44], and numerous studies have investigated the influence of dietary lipids on blood cholesterol levels[46–48]. Although there are still unresolved questions, it now seems probable that most prevalent polyunsaturated fatty acid (linoleic acid) has a cholesterol-lowering effect, whereas of the saturated fatty acids only myristic acid consistently increases serum cholesterol levels of normocholesterolemic people[45]. In hypercholesterolemic individuals with compromised LDL receptors, palmitic acid contributes to hypercholesterolemia. Monounsaturated fatty acids are neutral, but can appear equivalent to polyunsaturated when the exchange for linoleic acid is above limiting threshold for that acid[45,49,49a].

Early human studies indicated that palm oil can reduce the total serum cholesterol level in individuals with hypercholesterolemia[50,51] but it tends to have minimal effects in normocholesterolemic individuals[36,41,41a,42,45]. Brief summaries of the more recent, controlled studies are given below:

(1) A double blind cross-over study was carried out in Holland on 38 men[36]. In this trial 70% of the fat in a normal Dutch diet was replaced by palm oil. The following significant results were obtained.

a) No effect on serum total cholesterol
b) An 11% increase in HDL_2 cholesterol
c) An 8% decrease in the ratio LDL/HDL_{2+3}
d) A 9% decrease in LDL triglycerides

There was an associated increase in serum apolipoprotein A1 and decreases in apolipoprotein B and the apoB/apoA1 ratio.

It was concluded that the changes effected by the palm oil rich diet might slightly reduce the cardiovascular risk profile.

(2) In the same project plasma lipoprotein(a) was also measured[14]. There was a highly significant 10% decrease in Lp(a) during consumption of the palm oil rich diet. This result was consistent with those from two other independent laboratories (see later in this article[15,16]).

(3) In a study of 30 middle-aged men[37], six different fats were fed as ingredients in a normal American diet, forming 60% of the total fat intake. Fat represented 40% of the dietary energy. When palm oil was the test fat, total cholesterol was not affected but HDL-cholesterol (HDL-C) and apolipoprotein A1 increased, while apolipoprotein B decreased relative to baseline.

(4) Feeding nine men a diet containing 35% of the energy as fat, and one half of the fat as palm oil produced no change in plasma total or LDL-C, but a small rise in HDL-C occurred[38].

(5) Two trials were reported[39] using 21 and 30 subjects, respectively, in which half the dietary fat intake was supplied as potato crisps fried in the test oil. When palm olein was used there was a 10% increase in plasma HDL-C, which accounted for a 3% rise in total cholesterol.

(6) The effect of palm olein, used as the cooking oil in the preparation of food for 110 high school students, has been reported[40]. The food represented a typical Malaysian diet, with 35% of the energy content as fat. The palm olein rich diet did not significantly alter plasma total, HDL-C or LDL-C, but significantly increased the concentrations of apolipoprotein A1 (+11%) and apolipoprotein B (+9%). The ratio of Apo B to Apo A1 was not significantly affected. It was concluded that there was no adverse effect on plasma lipids.

(7) A study has been reported on 83 subjects consuming a Malaysian diet with 30% of the calorie intake as fat[41]. When palm olein formed 75% of the fat, the serum total cholesterol concentration was significantly lower than the level at entry (–9%), and the level on a diet with coconut oil (–20%). LDL-C and HDL-C levels were also lowered, and the LDL/HDL cholesterol ratio was reduced.

(8) Another study, carried out in 33 subjects, used a Malaysian diet with 34% fat calories[42]. When either palm olein or olive oil were fed as 23% energy (about 2/3 of the fat intake) neither fat resulted in a significant difference in serum total, LDL-C or HDL-C levels from those at entry. The data demonstrate the equivalent of palmitic and oleic acids in normal individuals.

The thromboxane/prostacyclin ratio in plasma was significantly reduced in the palm olein dietary period as compared with the olive oil period.

Palm oil and HDL-cholesterol
It is noteworthy that several of the studies summarized

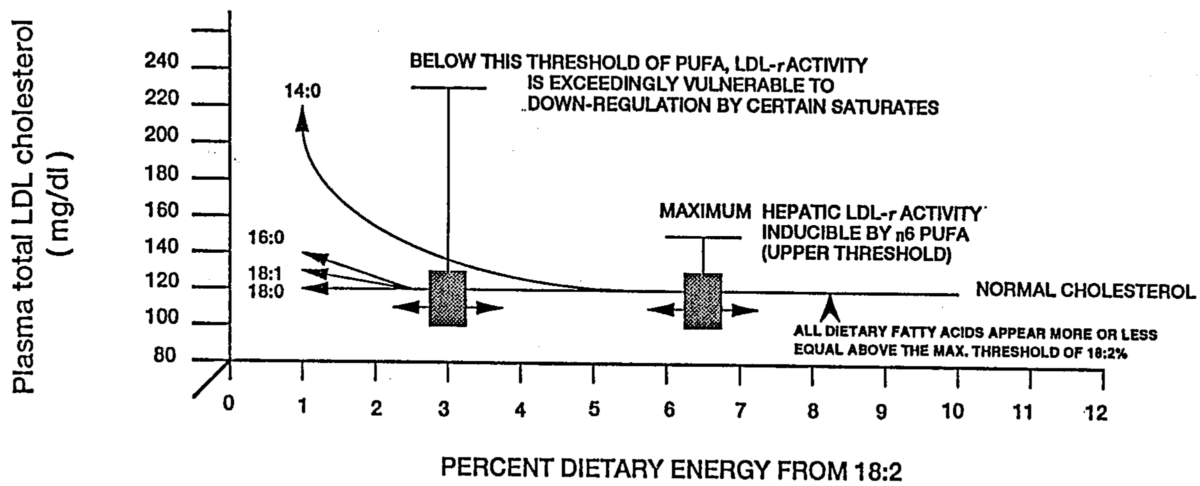

Figure 1. Dietary fatty acid threshold (source: Reference 56)

above(1–8) demonstrate the beneficial increase in HDL-C induced by palm oil.

Lipoprotein(a)

The level of lipoprotein(a), which is a potent indicator for coronary heart disease, was significantly reduced when palm oil provided most of the fat in an experimental diet[14].

Lipoprotein(a) is a distinct type of plasma lipoprotein that strongly predicts risk of cardiovascular disease. Its concentration in plasma seems to be largely genetically determined, but recently evidence has been obtained that it can be modulated by diet[14–16].

Reasons why palm oil does not raise plasma cholesterol

Results are now emerging which explain why palm oil, with its relatively high content of saturates, does not raise plasma cholesterol in the way that is usually expected. It is relevant that not all saturated fatty acids have the same effect on plasma cholesterol concentration[45,51], and that certain saturated fatty acids may even assist in removing and transforming excess of cholesterol from tissues to the liver.

Low-density lipoproteins transport cholesterol from the liver to the tissues, and in this form cholesterol promotes coronary heart disease[54]. LDL-C can also be taken up again by the liver, to be catabolized and excreted. This process is mediated by receptors on the surface of liver cells. For the synthesis of the LDL receptors, a specific messenger RNA is required. An increased production of this messenger RNA indicates a higher production of the LDL receptors. The more receptors are available, the better is the uptake of LDL-C by the liver and this may result in a reduction of the risk of coronary heart disease[52].

Another lipoprotein is also engaged in the 'reverse transport' of cholesterol from the tissues to the liver. This is the high-density lipoprotein, which is able to take up excessive cholesterol from peripheral tissues. The uptake of cholesterol is mediated by another 'target protein', apolipoprotein A1 (Apo A1). An increased concentration of Apo A1 in serum would indicate a higher amount of HDL particles and, consequently, an improved removal of excess cholesterol from peripheral cells and tissues. This cholesterol is subsequently transferred to other lipoproteins in plasma, and finally taken up by the liver, where it is catabolized; the products are then excreted[45–55b]. The observation by Lindsey and co-workers[53] that a palm oil diet fed to hamsters results in a significant increase in the production of both the messenger RNA for the LDL receptor and of Apo A1, therefore, points to two potential beneficial effects of palm oil in reducing cardiovascular risk.

Recently Hayes and co-workers[52] have also demonstrated that, in monkeys, dietary myristic acid (14:0) and palmitic acid (16:0) have very different effects on cholesterol metabolism, myristic acid being strongly cholesterolaemic. This effect was first noted in humans in 1965[46] but was subsequently largely ignored[45].

Hayes and Khosla[45,56] have advanced an hypothesis to explain the differing effects, reported in the literature over hree decades, of dietary fatty acids on plasma total cholesterol (TC). It is proposed that: (a) linoleic acid (18:2n-6) 'upregulates' LDL receptors (ie permits full activity), allowing LDL-C to be cleared from plasma, while myristic (14:0) acid 'down-regulate' the receptors (ie lower receptor activity) resulting in a rise in LDL-C, while (b) 12:0, 16:0 are equal and neutral on the cholesterolemic individuals and requirement for 18: 2 depends on the amount of 14:0 present. Above a threshold of 5–6% energy as 18:2, fatty acids of any kind (except 14:0) have minimal effects. Between 3% and 6.5% E as 18:2, 14:0 is the only fatty acid to increase plasma LDL-C, while below 3% E. 14:0 is highly hypercholesterolaemic and 16:0 only moderately so (Figure 1). These interactions may be further modified by (i) the quantity of cholesterol in the diet – at increasing levels, the sensitivity to saturated fatty acids may be greater, and by (ii) the initial concentration of plasma TC, ie subjects who are already hypercholesterolaemic may be more sensitive because their LDL receptors are saturated or down-regulated[45].

The hypothesis, based on initial results with monkeys, has been strengthened by reanalysis of published human data by the same authors, and there appears to be a good 'fit' with earlier data[45].

This provides an example of the important nutritional principle of nutrient balance, in this case the balance between different dietary fatty acids and cholesterol.

Palm oil and the prostacyclin/thromboxane balance

In a rat model of arterial thrombosis dietary palm oil performed comparably with other more unsaturated oils[57].

The risk of arterial thrombosis is at least partly determined by the potential of blood platelets to synthesize a pro-thrombotic compound, thromboxane, and of the arterial wall to generate the anti-thrombotic substance prostacyclin[58]. There is good evidence that palm oil reduces the tendency for thrombi to form in arteries, and that this is mediated by the ability of palm oil to promote a favourable shift in the balance between these substances. This has been demonstrated in rats[58–61] and also in rabbits[62].

Possible anti-tumour action of palm oil

Experimental work on rats, comparing palm oil in the diet with a number of other edible oils, showed that palm oil reduced the number of chemically induced tumours.

After induced carcinogenesis rats fed palm oil at 20% of the diet had significantly fewer tumours after 50 months than those fed the same level of corn or soya bean oil[63].

In a somewhat earlier study rats fed palm oil at 20% of the diet (before induction of carcinogenesis) showed no greater level of cancer than the 'controls' on a 5% corn oil diet. On the other hand, rats fed 25% beef fat or lard showed enhanced breast cancer development[64].

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